



DETECTION OF TRICHOMONAS TENAX (T. TENAX) IN BAD ORAL HYGIENE SAUDI PATIENTS IN TAIF CITY

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ABSTRACT

Gingivitis affects an estimated 80% of the population, most periodontal disease arises from or is aggravated by accumulation of plaque, and periodontitis is associated particularly with anaerobes such as Bacteroides. Calculus (tartar) may form calcification of plaque above or below the gum line, and the plaque that collects on calculus exacerbates the inflammation. The inflammatory reaction is associated with progressive loss of periodontal ligament and alveolar bone. T. tenax is a widespread flagellated protozoan that inhabits the human oral cavity in and around diseased teeth and gums. The role of Trichomonas tenax as a pathogen had been clearly implicated in various pathological processes that arise outside the boundaries of the mouth. Although a relationship between the increased occurrence of this protozoan and progression of periodontal disease has been demonstrated so the present study aimed to estimate the occurrence of T. tenax in individuals having oral infections. Calculi samples were collected from 58 patients who were diagnosed as having periodontitis and/or gingivitis, then were subjected to direct smear examination. The results showed that the positivity rate of T. tenax is 13%.

Conclusions: This result support the association between T. tenax infection and bad oral hygiene and suggest the usefulness of elimination of this protozoa to achieve radical cure of gingivitis and periodontitis

KEY WORDS: Trichomonas tenax, periodontitis, gingivitis

1. INTRODUCTION :

Periodontal disease: is two categories, gingivitis and periodontitis. Gingivitis is the primary, reversible stage of the disease process in which the inflammation is confined to the gingival (Merin, 2006). This inflammation is formed by plaque or bacteria and may be solve the problem with a thorough dental prophylaxis and consistent home care (Grove, 1982). Periodontitis is the later phase of the disease process is known as inflammatory disease of the deeper supporting structures of the tooth (periodontal ligament and alveolar bone) caused by microorganisms (Novak, 2006). This inflammation results the progressive destruction of these tissues, leading to gingival slump, periodontal pocket formation, or both. can be reduced or eliminated from mild to moderate periodontal pockets by proper removal of plaque and calculus (Carranza and Takei, 2006). Although bone loss is irreversible, it is possible to arrest its progression (Merin, 2006).

Most periodontal disease generate from, accumulation of plaque, and periodontitis is related particularly with anaerobes such as Porphyromonas gingivalis, Bacteroides forsythus, and Actinobacillus actinomycetem comitans. Calculus (tartar) may form from calcification of plaque above or below the gum. The inflammatory reaction is associated with progressive loss of periodontal ligament and alveolar bone and, eventually, with mobility and loss of teeth. Periodontal diseases are ecogenetic in the sense that, subjects susceptible by genetic or environmental factors (such as polymorphisms in the gene for interleukin 1, cigarette smoking, immune depression, and diabetes), the infection leads to more rapidly progressive disease. Osteoporosis also appear to have some impact on periodontal bone loss. The possible effects of periodontal disease on systemic health, via pro-inflammatory cytokines (Coventry et al., 2000). With plaque formation bacteria from healthy sites consist of predominately non motile, Gram positive, aerobic facultative rods and cocci. Gingivitis is caused by an increase in the gross numbers of bacteria, and this raise is primarily of motile Gram-negative rods and anaerobic species (Quirynen et al., 2006). In established periodontal disease, Gram-negative rods account for about 74% of the microbiota flora. high numbers of spirochetes are found in almost all periodontal pockets, and anaerobic organisms compose 90% of the bacterial species in chronic periodontal disease (Slots, 1979).

Chronic gingivitis to some degree affects over 90% of the population. the prognosis is good if treated, but otherwise it may progress to periodontitis and tooth mobility and loss. Marginal gingivitis is painless but may appear with bleeding from the gingival crevice, especially when brushing the teeth. The gingival margins are slightly red and swollen, eventually with moderate gingival hyperplasia. Management—Unless plaque is assiduously removed and preserved under control by tooth brushing and flossing and, where necessary, by removal of calculus by scaling and polishing by dentist, the condition will recur. Although gingivitis has a bacterial component, systemic antimicrobials have benefit. Surgical reduction of hyperplastic tissue by a periodontist (gingivectomy and gingivoplasty) may occasionally be required (Coventry et al., 2000).

The bacteria in the subgingival plaque secrete toxins as well as metabolic products such as cytotoxins and bacterial endotoxins, which can invade tissues on their own and in turn cause inflammation to the gingival and periodontal tissues. this inflammation causes damage to the gingival tissues and initially results in gingivitis, white blood cells and other inflammatory mediators migrate out of the periodontal soft tissues and into the periodontal space due to increased vascular permeability and increased space between the crevicular epithelial cells. When released into the sulcus, these enzymes will cause further inflammation of the delicate gingival and periodontal tissues, it is the host response that often damages the periodontal tissues, as periodontal disease progresses over time, the attachment loss continues in a nonlinear pattern as active stages of destruction are followed by quiescent phases (burst). The end stage of periodontal disease is tooth loss, however, the disease has created significant problems before tooth exfoliation (Nisengard et al., 2006).

The oral cavity of human is conquered through bacteria, fungi and protozoa, between these microorganisms, Trichomonas tenax is generally found in the oral cavity and bad oral hygiene patient and periodontal disease included. The development of T. tenax is rely upon host age. Different determinants are responsible for transporting parasite that involve incidence by saliva through kissing, or use of dirty dishes and drinking water. Depending on oral health status, level of infection is recorded between 0 to 25%. Trichomonas tenax is an flagellated protozoan that inhabits the human oral cavity in and over diseased teeth and gingiva. Its predominance in the mouth varies from 4% to 53% and it is believed to be a commensal (Ghabanchi et al., 2010). It is suspected in different infection outside the limits of the oral cavity. It has been discovered in a submaxillary gland infection. T. Tenax is familiar as common parasite of oral cavity and also discovered in submaxillary glands infra-auricular lymph node infection, pleural and respiratory fluids in cases of pleuro-pulmonary infection. This protozoan is categorized in same genus as Trichomonas vaginalis. T. vaginalis and T. tenax have extraordinary genetic identity and that T. vaginalis has higher levels of gene expression when compared to that of T. tenax. The information may recommend that T. tenax might be a variant of T. vaginalis. When in fact the pathogenicity of T. vaginalis has been comprehensively studied while little is known about the pathogenic role of T. tenax. So this work is aimed to estimate the detection rate of T. tenax in patients having bad oral hygiene.

2. SUBJECTS AND METHODS

Subjects

Cross section study were conducted on 58 patients of both sexes who attended dental out-patients' clinics in Taif. In the period from January 2017 to April 2017

Selection criteria

The selected patients were suffering from periodontitis or gingivitis as established by a periodontist. Each of these patients were subjected to a full history taking, radiographic and clinical observation of their mouths (periodontal pocket, calculus and debris, color, swelling and change in consistency of gingiva).

Exclusion criteria

The patients receive antibiotic or periodontal treatment.

Sampling and materials

58 Periodontal swab samples were taken from patients and rapidly spread on glass slide then they were fixed with ethanol as preservative after that the fixed samples were transferred to our university lab to subjected to Gram stain . After that the samples were subjected to the microscopic examination.

Microscopic examination

The samples were examined with x40 and x100 objective of light microscope - Statistical Analysis:

Data were coded, entered, and analyzed using SPSS version 19 .

Ethical considerations:

Ethical approval for this study was obtained from the Ethics Review Committee of the College of Applied Medical Sciences at Al-Taif University. Moreover, all patients included in the study were informed of the study objectives and a written signed consent was taken from each one of them.

4. RESULT

Regarding sex distribution among samples the females were 34 (59%) however males were 24 (41%) as showing in figure (1). Based on microscopic examination a lot of variation was detected of microorganisms. Mixed samples Gram positive and negative were the most dominant by 24 (41%) samples while gram negative bacilli samples were the least by 2 (3%) samples and the results showed Gram positive cocci were 20 (34%), 5 (9%) Fungi , 7 (13%) positive T.tenax samples as showing in figure (2) . Some samples were recorded as photos in picture (1 and 2).

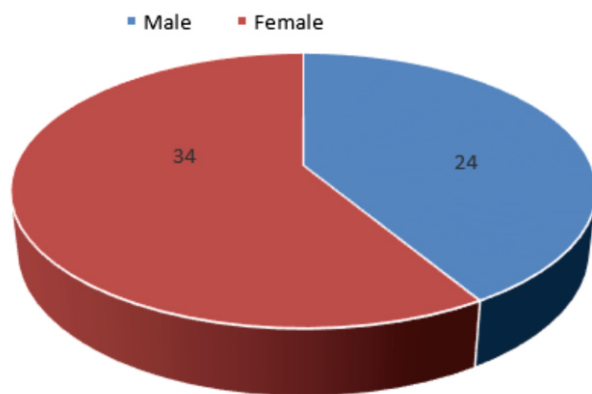


Figure (1) Male and Female distribution among the samples collection.

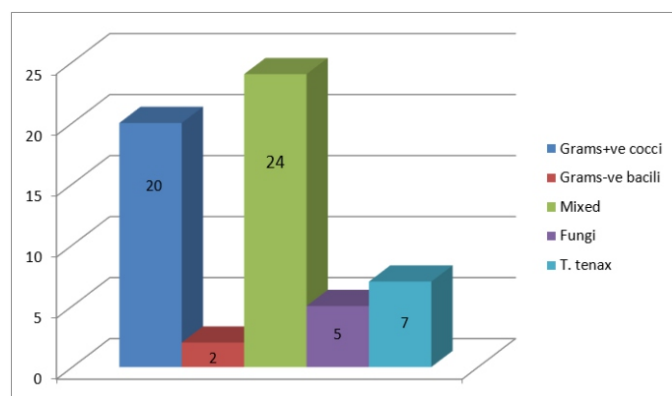
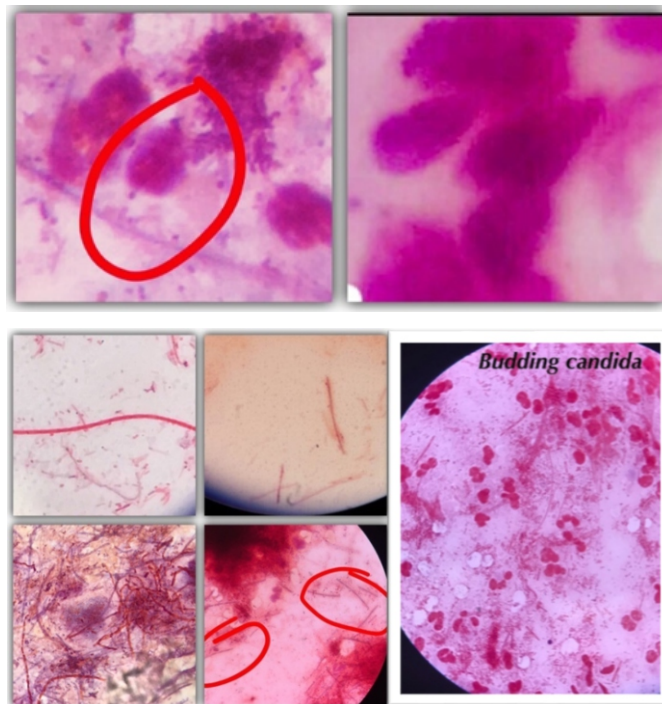
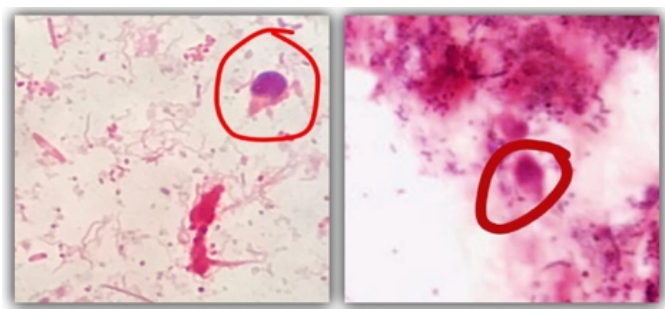


Figure (2): Number of different types of microorganisms detected in the study group.



Picture(2) Different types of fungi under the microscope.

4. DISCUSSION

The human oral cavity is home to numerous microorganisms (Feki et al.,1990). Since mouth infection symptoms derive from the interaction between pathogenic microbiota and the host's defense mechanisms, it is extremely important to study the microorganisms that cause periodontal disease in adults. Hence, local irritating factors, particularly the dental bacterial biofilm, seem to have a critical role in the susceptibility to, and onset and progression of, periodontal disease (Nocito et al.,2000). Gingivitis is a form of periodontal disease. Periodontal disease involves inflammation and infection that destroys the tissues that support the teeth, including the gums, the periodontal ligaments, and the tooth sockets (alveolar bone). Gingivitis is due to the long-term effects of plaque deposits. Plaque is a sticky material made of bacteria, mucus, and food debris that develops on the exposed parts of the teeth. It is a major cause of tooth decay. If you do not remove plaque, it turns into a hard deposit called tartar that becomes trapped at the base of the tooth. Plaque and tartar irritate and inflame the gums. Bacteria and the toxins they produce cause the gums to become infected, swollen, and tender. This exposes people to infection with *Trichomonas tenax* Causing inflammation of the gums. The oral cavity is suitable for invasion of many microorganism, the protozoan *Entamoeba gingivalis* and *Trichomonas tenax* are recognized eukaryotic representatives. Although these forms are not generally associated with pathogenesis, their presence in the oral cavity is taken as a sign of poor dental hygiene (Jian et al.,2008 and Abulqumsaan et al., 2010).

It is extremely important to study the microorganisms that cause periodontitis and gingivitis in human (Junior et al.,2011). *Entamoeba gingivalis* and *Trichomonas tenax* were the first commensal found in human oral cavity, they occur only as a trophozoite, and these are found in gingival tissues, particularly in suppurative, inflammatory processes, due to there are preference for anaerobic environments (Chen et al.,2001).

It is believed that these commensal could be opportunistic, that these, capable of proliferating in a gingival environment modified by periodontal and gingivitis disease (El-Azzoumi et al.,1994). The trophozoites of *Trichomonas tenax* are most probably transmitted from person to person by close contact,since they exhibit only slight resistance to the environment . Kissing may be play a role in transmission, but direct passage by many ways such as mutual usages of cups, spoon, fork, and subjects contaminated by an infected person probably is a mode of transmission also (Al- Najar and Adnan 1986).

There are only few reports on the role of oral commensals in the pathogenesis of periodontitis and gingivitis despite the high incidence of certain protozoa, such as *Trichomonas tenax* . study was conducted in Iran on *Entamoeba gingivalis* (*E. gingivalis*) and *Trichomonas tenax* (*T. tenax*) may be responsible for oral parasitic infection (Ghabanchi et al.,2010), the case control study conducted in Iran showed that in the case group, nine patients were infected to the parasites, six (66.7%) with *E. gingivalis*, and three (33.3%) with *T. tenax* . This study showed that patients with periodontal disease were more prone to parasitic infections. The prevalence of oral trichomoniasis in study (33.3%) was compatible with many other published reports, which mostly ranged from 12 - 32% (Sarowaska et al.,2004).

The results of our research were on 58 patients, 34(59%) women and men 24 (41%) and their ages ranged from 20 to 50 years . 34% had Gram positive cocci and 3% Gram negative bacilli . Where the mixture is between Gram positive cocci and Gram negative bacilli was 41% Of samples . the sample contained fungi was 9% . In the current study the frequency of oral trichomoniasis in patients having oral infections 13% is compatible with many published reports in which it mostly has ranged from 12% to 32% . Among them was suffering from gingivitis. Patients did not use antibiotics. The prevalence of *T.tenax* in our study is 13% this was lower than the previous study in Iran 33.3% . Many factors might have affected the differences reported; including sample size of the patients examined, the methods of parasite detection used and the type of samples analyzed . As parasitic infections are relatively common in our patients with periodontal disease and increase in periodontal disease would result into an increase in parasitic infestations, it seems that following oral and general hygiene instructions are essential in control of parasitic infestations. So this result support the association between *T. tenax* infection and bad oral hygiene and suggest the usefulness of elimination of this protozoa to achieve radical cure of gingivitis and periodontitis, we recommend to do test for the diagnosis of *T.tenax* to decrease the resistance to the treatment

REFERENCES:

- Abulqumsan, T. and Yolasigmaz, A. (2010). The investigation of *Entamoeba gingivalis* and *Trichomonas tenax* in a group of patients with periodontal disease. *Turkey Society for Parasitology* , 34(2), p. 91-4.
- Al- Najar, S. and Adnan, E. (1986). The first record of *Entamoeba gingivalis* in Iraqi patients. *J Fac Med Baghdad*, 28 (2), p. 73-80.
- Bellanger, A., Cabaret, O., Costa, J., Foulet, F., Bretagne, S., Botterel, F. (2008). Two unusual occurrences of trichomoniasis: rapid species identification by PCR. *J Clin Microbiol*.
- Carranza, F. and Takei, H. (2006). Rationale for periodontal treatment. *Carranza's Clinical Periodontology* St Louis WB Saunders.p.630-635.
- Chen, F., Wen, R., Liu, Y., Chen, L., Lin, G. and Hong, Y. (2001). Studies on periodontal disease caused by *Entamoeba gingivalis* and its pathogenic mechanism. *Rev China Med J* . 114(12), p. 12-15.
- Coventry, J., Griffiths, G., Scully, C., Tonetti, M. (2000). ABC of oral health Periodontal disease Periodontitis Gingival bleeding Gingival ulcers. P. 321.
- Duboucher, C., Farto-Bensasson, F., Chéron M., Peltier, Y., Beauvils, F. and Périé, G. (2000). Lymph node infection by *Trichomonas tenax*: Report of a case with co-infection by *Mycobacterium tuberculosis*. *Hum Pathol* .
- El-Azzoumi M. and, Al-Brady M. (1994). Frequency of *Entamoeba gingivalis* among periodontal and patients under chemotherapy. *J Egypt Soc Parasitology* , 24(3), p. 649-55.
- Feki, A. and Molet, B. (1990). Importance of *Trichomonas tenax* and *Entamoeba gingivalis* protozoa in the human oral cavity. *Rev Odontostomatol*. 19(1), p. 37-45.
- Ghabanchi, J., Zibaei, M., Afkar, M., Sarbazie, A. (2010). Prevalence of oral *Entamoeba gingivalis* and *Trichomonas tenax* in patients with periodontal disease and healthy population in Shiraz, southern Iran. *Indian Journal of Dental Research*. 21 (1), p. 89.
- Grove, K. (1982). Periodontal disease. *The Compendium on Continuing Education* 564-570.
- Jian, O., Kolansky, S. and Baloach, W. (2008). *Entamoeba gingivalis* pulmonary abscess-diagnosed by needle aspiration. *Cyto J*. 5(12).
- Junior, L., Melo, C., Santana, W. and Ribeiro, J. (2011). Incidence of *Entamoeba gingivalis* and *Trichomonas tenax* in samples of dental biofilm and saliva with periodontal diseases. *RGO-Rev Gaucha Odontol* proto Alegre . 59 (1), p. 35-40.
- Kamel, A., Rouetbi, N., Chakroun, M. and Battikh M. (1996). Pulmonary eosinophilia due to *Trichomonas tenax*. *Thorax*.
- Kucknoor, S., Mundodi, V. and Alderete, J. (2009). Genetic identity and differential gene expression between *Trichomonas vaginalis* and *Trichomonas tenax*. *BMC Microbiol*. 9, P.58.
- Merin, L. (2006). Results of periodontal treatment. *Carranza's Clinical Periodontology* St Louis WB Saunders. P.1206-1214.
- Mahdi, K. and Al-Saeed A. (1993). "Trichomonas Tenax In Basrah, Iraq."
- Nisengard, J., Kinder, S., Newman, G. and Miyasaki, T. (2006). Microbial interactions with the host in periodontal diseases. *Carranza's Clinical Periodontology* St Louis WB Saunders.p. 228-250.
- Nocito, I., Vasconi, D., Ponce, L. and Horianski, P. (2003). *Entamoeba gingivalis* and *Trichomonas tenax* in diabetic patients. *RCOE*. 8(1).p. 13-23.
- Novak, J. (2006). Classification of diseases and conditions affecting the periodontium. *Carranza's Clinical Periodontology* St Louis WB Saunders.p. 100-109.
- Prole, L. and Taylor, W. (2011). Identification of Intracellular and Plasma Membrane Calcium Channel Homologues in Pathogenic Parasites. Moreno SN, editor. *PLoS One*. 6(10).p. 218.
- Quirynen, M., Teughels, W., Kinder, S. and Newman, G. (2006). Microbiology of periodontal diseases. *Carranza's Clinical Periodontology* St Louis WB Saunders.p. 134-169.
- Roberts, L. (2009). *Foundations of Parasitology*. 6th ed. McGraw-Hill, p. 95-97.
- Sarowaska, J., Wojnicz, D., Kaczowski, H. and Jankowski, S. (2004). The occurrence of *Entamoeba gingivalis* and *Trichomonas tenax* in patients with periodontal disease, immunosuppression and genetic diseases. *Adv. Clin. Exp. Med*. 13.p. 291-297.
- Slots, J. (1979). Subgingival microflora and periodontal disease. *J Clin Periodontol*. 6 (5).p. 351-382.
- Yamamoto A, Hashimoto T, Asaga E, Hasegawa M, Goto N. Phylogenetic Position of the Mitochondrion-Lacking Protozoan *Trichomonas tenax*, Based on Amino Acid Sequences of Elongation Factors 1 α and 2. *J Mol Evol*. 1997 Jan 13;44(1):98-105.